

From 'Unexplained' Necrotic Fingertip Wounds to Lupus: An Atypical Secondary Raynaud Phenomenon in a Rural Kenyan Hospital

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Abstract

Raynaud's phenomenon (RP) is a transient, recurrent vasospastic disorder characterized by a triphasic discoloration of the fingertips manifesting as pallor, cyanosis, and erythema. Some cases may be associated with necrosis of the fingertips due to digital ischemia. RP may be primary (idiopathic) or secondary, with the latter due to many causes, especially autoimmune diseases (e.g., scleroderma and lupus). A diagnosis of RP is based on a detailed history and nailfold capillaroscopy using an ophthalmoscope, a dermatoscope, and a video capillaroscope. In primary RP the capillaroscopy is normal, while secondary RP shows abnormal vascular changes with specific patterns for scleroderma and lupus. Lupus patients with RP tend to have a less severe clinical course of the lupus compared to those without RP. In this study, we present how using an ophthalmoscope for nailfold capillaroscopy led to the diagnosis and management of secondary Raynaud's phenomenon (RP) caused by systemic lupus erythematosus (SLE) in a young woman from Kenya.

Keywords: Raynaud's phenomenon, nailfold capillaroscopy, systemic lupus erythematosus, Kenya.

1. INTRODUCTION

Raynaud's phenomenon (RP) is a vasospastic disorder of the finger tips due to recurrent, transient, and reversible vasoconstriction of the peripheral blood vessels when exposed to cold or emotional distress (1).

RP is characterized by a triphasic discoloration of the affected parts, often beginning with pallor (white attack), followed by cyanosis (blue

attack), and ending with erythema (reperfusion) (1). The changes may last from several minutes to hours. RP may be idiopathic (primary RP, occurring in about 80-90% of individuals) or caused by underlying diseases, especially other autoimmune diseases like systemic sclerosis (most common), systemic lupus erythematosus (SLE), etc. (secondary RP, occurring in 10-20% of patients). See Table 1 below for secondary causes of RP, adopted from Haque and Hughes (2).

Table 1. Secondary causes of RP (2)

Vascular (usually proximal large vessel disease, often unilateral symptoms)	Compressive (e.g., cervical rib) Obstructive (e.g., atherosclerosis); inflammatory vascular disease (e.g., thromboangiitis obliterans (Buerger's disease))
Occupational	Hand-arm-vibration syndrome (vibration white finger)
Autoimmune conditions	Systemic sclerosis, systemic lupus erythematosus Sjogren's syndrome, mixed connective tissue disease/overlap syndromes, undifferentiated connective tissue disease, idiopathic inflammatory myopathies
Drug-/chemical-related	Amphetamines, beta-blockers, bleomycin, cisplatin, clonidine, cyclosporine, interferons, methysergide, polyvinyl chloride
Conditions associated with increased plasma viscosity and reduced digital perfusion	Cryoglobulinaemia, cryofibrinogenaemia, paraproteinaemia, malignancy (including as a paraneoplastic phenomenon)
Other causes and associations	Carpal tunnel syndrome, frostbite, hypothyroidism

Digital ischemia causes pain, which may fully resolve or be associated with digital necrosis, especially in secondary RP (2). A diagnosis of RP is based on a detailed history, direct observation, and nailfold capillaroscopy. The latter can be done using an ophthalmoscope, a dermatoscope, a stereomicroscope, a conventional optical microscope, and a video capillaroscope (3). The use of an office ophthalmoscope is non-invasive, inexpensive, portable, and easy to learn and use (4). It has yielded 4 well-described patterns,

including a normal pattern, a scleroderma pattern, a lupus pattern, and a non-specific pattern (5). In primary or idiopathic RP, the capillaroscopy is normal, and patients do not require further extensive investigations.

In secondary RP, capillaroscopy is abnormal, including giant and bushy capillaries, hemorrhages, and avascular areas (4). Figure 1 below by Meinema et al. captures some capillaroscopy images in patients with SLE (6).

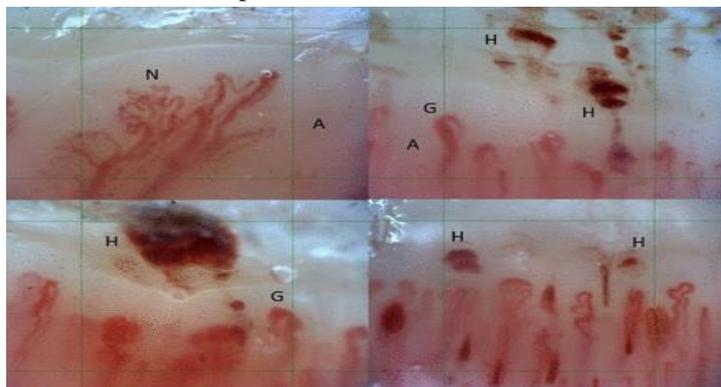


Figure 1. Capillaroscopy patterns in patients with SLE showing giant capillaries (G), hemorrhages (H), and neovascularization (abnormal shapes) (N), with avascular areas (A) (6)

RP in SLE has been described in the literature. An earlier prospective cohort study of over 220 SLE patients showed a 40% prevalence of RP in SLE. This finding was associated with a less severe course of SLE (specifically a lesser incidence and severity of lupus nephritis) among the patients who had RP (7). A 2015 study suggested that RP occurred in SLE patients who were older and seemed to have a lower prevalence of glomerulonephritis, hemolytic anemia, and serositis (8). A 2022 prospective cohort study of over 480 SLE patients showed that more than a third had RP, which was negatively associated with hemolytic anemia and glomerulonephritis (9). This evidence has led to the conclusion that the occurrence of RP in patients with SLE is associated with milder disease and a favorable prognosis of the SLE. The current approach to the management of RP involves the avoidance of cold, stress management, use of calcium channel blockers (or phosphodiesterase-5 inhibitors and topical nitrates) and prostacyclin analogues (e.g., iloprost), and the management of any secondary causes of RP as per guidelines (10).

2. CASE SUMMARY

2.1. History and Physical Examination

A 19-year-old nulliparous lady from Narok town, Kenya, a recent high school leaver with no

known personal or family history of chronic medical conditions, first presented to us in late January 2024 with a 3-month history of painful fingertip wounds involving both hands. These wounds occurred spontaneously when her fingertips were exposed to cold temperatures, such as while washing dishes in the morning. The fingertips would turn whitish, numb, and painful, and later on greyish, before regaining their pink color after about 3-5 minutes, or sooner when dipped into warm water. Over time, she developed painful cracking wounds over the fingertips with associated numbness and inability to clench fists. She had tried using local herbal treatment with no success. She had no history to suggest obvious lupus (no joint pains or swelling, oral ulcers, skin rashes, alopecia, recurrent infections, etc.) or overt heart disease. She had observed progressive facial swelling over the past 2 weeks, but she had no previous history of recurrent throat or skin infections, nor any signs of pedal edema, hematuria, or hypertension that would suggest nephritic syndrome.

On examination, she was a healthy-looking young woman with obvious facial puffiness and a resting tachycardia of 102 bpm, BP 115/72 mmHg, SPO2 of 96% in room air, and a weight of 77 kg. Her hands and fingers were normal-looking but she had pulp ulceration and necrosis involving the second and third fingertips on both

hands. See figure 2 below. She had a normal joint and musculoskeletal examination. She had no edema, skin rashes, oral ulcers, or alopecia. Her cardiovascular examination showed no stigmata of infective endocarditis with no splinter hemorrhages, a normal S1 and S2 with no murmurs, and she was not in heart failure. She had no clinical features of scleroderma (no syndactyly, calcinosis cutis, sandpaper skin rash,

and she had a normal mouth aperture with no microstomia).

Her abdominal, respiratory, and neurological examinations were normal. A bedside capillaroscopy using an ophthalmoscope with KY jelly revealed tortuous dilated capillary loops most visible in the 1st to 4th fingernails on the right hand and the 2nd to 4th fingernails on the left hand.



Figure 2. Shows ulceration and necrosis of the 2nd and 3rd fingertips on both hands.

2.2. Work-Up, Management, and Follow-Up

She had a normal complete blood count, which included a WBC of $6.8 \times 10^3/\mu\text{L}$, an Hb of 12.4 g/dL, and platelets of $410 \times 10^3/\mu\text{L}$; a normal urinalysis showing no proteinuria or hematuria and a negative pregnancy test; a normal random blood glucose level of 108 mg/dL; and a total cholesterol level of 162 mg/dL.

She was HIV negative, she had an ESR of 40 mm/hr, a negative rheumatoid factor, a normal chest x-ray, a normal EKG, and a normal POCUS echocardiogram.

We made a clinical diagnosis of secondary Raynaud phenomenon (RP), most likely due to an autoimmune disease (in view of the abnormal nailfold capillaroscopy and on epidemiological basis), with the main consideration being systemic lupus erythematosus (SLE). Scleroderma was unlikely in the absence of syndactyly, calcinosis cutis, sandpaper skin rash, or upper gastrointestinal symptoms. We did an interval serology, which was positive for anti-nuclear antibodies (ANA) and anti-double stranded DNA (ds-DNA). See figures 3 and 4 below.

Test	Result	Units	Flag	Ref Interval
ANTI NUCLEAR ANTIBODIES (ANA)				
Result	Weak positive			Negative (<1:100)
Pattern	Homogeneous nucleolar			
Grade	+			
Estimated titre	1:100			
Interpretation Guidelines (sample screening dilution - 1:100)				
Negative (<1:100): No immunofluorescence				
+: Weak positive (1:100)				
++: Moderate positive (1:320)				
+++: Strong positive (1:1000)				
++++: Very strong positive (1:3200)				
Test Description:				
Antinuclear antibodies (ANA) are detected using an indirect immunofluorescence slide assay test, HEp-20-10 substrate. The ANA test is a sensitive screening test used to detect autoimmune diseases as one parameter in a multi-criteria diagnostic process encompassing both clinical & lab criteria. ANA lacks specificity and is associated with a variety of autoimmune, infectious, inflammatory conditions, cancers & drugs. It may occur in healthy individuals, prevalence increasing with age. The pre-test probability/ clinical context therefore affects interpretation.				

Figure 3. A positive anti-nuclear antibody test with a titer of 1:100

Test	Result	Units	Flag	Ref Interval
ANTI DOUBLE STRANDED DNA (DS-DNA ANTIBODY)	Positive			Negative <1:10 titer
Test Description:				
Anti double-stranded DNA is tested using an indirect immunofluorescence slide assay test, Crithidia luciliae substrate. Anti ds-DNA test is used in evaluation of SLE as one parameter in a multi-criteria diagnostic process encompassing both clinical & lab criteria.				
FLAG KEY: L= Low; H= High; *= Abnormal; != Critical or notifiable result				

Figure 4. A positive anti-double-stranded DNA test

We refined our diagnosis to secondary RP due to SLE and put her on nifedipine (for the RP), hydroxychloroquine, prednisone (started at a dose of 40 mg daily and tapered off over two months), methotrexate with folate, furosemide, atorvastatin, and supportive care, including vitamin D-calcium combo tablets (for osteo-protection), omeprazole (gastro-protection), fingertip wound dressing with antibiotic topical creams, gabapentin for peripheral neuropathy, warm clothing with woolen gloves and stockings, contraception counselling, etc. On follow-up, she developed photosensitivity, oral ulcers, and polyarthralgia at different times, which fully responded to treatment. She has remained stable while taking adjusted doses of her medications, which have resulted in very low SLE disease activity index (SLEDAI) scores; however, she experiences occasional mild to moderate flare-ups due to poor drug compliance. The necrotic fingertips have fully healed, and the RP subsided after 2 months of treatment. The ESR has remained elevated between 30 and 55 mm/hr. The vitals have been normal, and the weight has been maintained between 77 and 79 kg. There has been no evidence of nephritis (all urinalyses to date have had no proteinuria, hematuria, sediments, or casts), hemolytic anemia, serositis, neurolupus, or any other severe manifestations of SLE.

3. DISCUSSION

The typical RP presents with a triphasic discoloration of the fingertips involving pallor, cyanosis, and erythema (1). Our patient presented with an atypical pattern that included pallor associated with numbness and pain, greyish discoloration (most likely due to her dark African skin), and erythema on rewarming. This was associated with progressive painful necrosis of the fingertips. We were concerned about an autoimmune etiology from the onset given her demographics and the finding of dilated, tortuous capillaries on a simple office nailfold capillaroscopy (4) using a hand-held ophthalmoscope and KY jelly. An abnormal nailfold capillaroscopy in RP is consistent with secondary RP, with the main differential diagnoses of systemic sclerosis (scleroderma) and lupus (5). Primary RP usually has a normal capillaroscopy. Our patient had no stigmata of scleroderma, and further serological tests (a positive ANA, anti-ds-DNA, and a high ESR), as well as the evolution of other symptoms on follow-up (photosensitivity, oral ulcers, and polyarthralgia), are consistent with and fulfill the 2019 European League Against Rheumatism/

American College of Rheumatology (EULAR/ACR) criteria for SLE (11). The patient has responded to treatment with hydroxychloroquine, methotrexate, steroids, and supportive care, leading to clinical remission for long periods of time. So far, there has been no evidence of lupus nephritis or renal failure despite a positive anti-ds-DNA test, which is a specific SLE marker and is considered to be an indicator of lupus nephritis (12). Additionally, there have been no other manifestations of severe lupus, like hemolytic anemia, neuro-lupus, or pulmonary hemorrhage syndromes. This relatively mild course of SLE aligns with studies suggesting that the occurrence of RP in SLE is associated with a less severe disease phenotype (7, 8, 9). The main learning point in this case is that the finding of an abnormal nailfold capillaroscopy, performed with a simple office ophthalmoscope during the evaluation of atypical RP, has led to the diagnosis and effective management of SLE as a secondary cause of RP.

4. CONCLUSION

The evaluation of RP should begin with a nailfold capillaroscopy to distinguish between primary RP (in which capillaroscopy is normal) and secondary RP (in which capillaroscopy is abnormal). Systemic sclerosis and SLE are the most common causes of secondary RP. The occurrence of RP in SLE seems to be associated with a less severe SLE disease clinical course.

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ETHICAL COMPLIANCE

Informed consent was obtained from the patient for this publication.

REFERENCES

- [1] Nawaz I, Nawaz Y, Nawaz E, Manan MR, Mahmood A. Raynaud's Phenomenon: Reviewing the Pathophysiology and Management Strategies. *Cureus*. 2022; 14(1): e21681.
- [2] Haque A, Hughes M. Raynaud's phenomenon. *Clin Med (Lond)*. 2020; 20(6):580-7.
- [3] Smith V, Ickinger C, Hysa E, Snow M, Frech T, Sulli A, et al. Nailfold capillaroscopy. *Best Practice & Research Clinical Rheumatology*. 2023; 37(1):101849.
- [4] Anders HJ, Sigl T, Schattenkirchner M. Differentiation between primary and secondary Raynaud's phenomenon: a prospective study

- comparing nailfold capillaroscopy using an ophthalmoscope or stereomicroscope. *Annals of the Rheumatic Diseases*. 2001; 60(4):407-9.
- [5] Minkin W, Rabhan NB. Office nail fold capillary microscopy using ophthalmoscope. *J Am Acad Dermatol*. 1982; 7(2):190-3.
- [6] Dieneke S-M, Sandy CB, Amara Nassar-Sheikh R, Mariken PG, Maritza AM-H, Wineke A, et al. Nailfold capillary scleroderma pattern may be associated with disease damage in childhood-onset systemic lupus erythematosus: important lessons from longitudinal follow-up. *Lupus Science & Medicine*. 2022; 9(1):e000572.
- [7] Dimant J, Ginzler E, Schlesinger M, Sterba G, Diamond H, Kaplan D, et al. The clinical significance of Raynaud's phenomenon in systemic lupus erythematosus. *Arthritis Rheum*. 1979; 22(8):815-9.
- [8] Heimovski FE, Simioni JA, Skare TL. Systemic lupus erythematosus and Raynaud's phenomenon. *An Bras Dermatol*. 2015; 90(6):837-40.
- [9] Barbacki A, Rached-d'Astous N, Pineau CA, Vinet E, Grenier LP, Kalache F, et al. Clinical Significance of Raynaud Phenomenon in Systemic Lupus Erythematosus. *J Clin Rheumatol*. 2022; 28(2):e488-e90.
- [10] Wigley FM, Flavahan NA. Raynaud's phenomenon. *New England Journal of Medicine*. 2016; 375(6):556-65.
- [11] Aringer M, Costenbader K, Daikh D, Brinks R, Mosca M, Ramsey-Goldman R, et al. 2019 European League Against Rheumatism/American College of Rheumatology Classification Criteria for Systemic Lupus Erythematosus. *Arthritis Rheumatol*. 2019; 71(9):1400-12.
- [12] Yung S, Chan TM. Mechanisms of Kidney Injury in Lupus Nephritis - the Role of Anti-dsDNA Antibodies. *Front Immunol*. 2015; 6:475.

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